

The University of Chicago

FACTORS INFLUENCING BRAIN
POTENTIALS DURING SLEEP

A DISSERTATION SUBMITTED TO THE FACULTY
OF THE DIVISION OF THE BIOLOGICAL SCIENCES
IN CANDIDACY FOR THE DEGREE OF DOCTOR OF
PHILOSOPHY

DEPARTMENT OF PHYSIOLOGY

1939

By
HELEN BLAKE

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HELEN BLAKE

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LOOMIS, HARVEY, AND HOBART (1937) have divided the period of sleep into several stages on the basis of changes in human brain potential patterns; Davis *et al.* (1938) have further analyzed the changes during the first part of the night; and Blake and Gerard (1937) have found delta wave intensity to parallel depth of sleep as determined by response to an auditory stimulus. Many bodily states show diurnal variations, for example: consciousness, movement, autonomic tone, skin resistance, and temperature (Kleitman, 1929). Some of these factors, as well as certain abnormalities in sleep, such as narcolepsy and sleep after experimental insomnia, have now been studied in relation to brain potentials in an attempt further to elucidate the mechanisms involved in the reversible change from wakefulness to sleep.

METHOD

Small silver (or solder) bipolar disc-electrodes, fastened to any two regions of the scalp (by collodion), were used to lead off the brain potentials in some experiments; more often monopolar leads were used consisting of a ring on the ear lobe and a disc on the vertex or occiput. Two independent, five-stage, resistance-capacity, push-pull amplifiers (time constant = 0.5 sec.; Offner, 1936) fed cathode ray oscillographs and crystographs (Offner and Gerard, 1937).

Depth of sleep was measured as before by the duration of a constant sound required to elicit a response from the subject. Movement was measured by a motility box (Kleitman, 1932) and by muscle potentials picked up in the head leads. The latter was a more sensitive index, the record consisting of periods of muscle tension rather than of gross movements, and sometimes gave indication of changes not detected by the motility box. Temperature was taken orally. The subjects were usually placed on a bed in a darkened room, quiet except for the constant hum of a motor; but in one series of narcoleptics the subjects sat in an upright position and with ordinary illumination. Twenty-two experiments were performed on 8 normal adult subjects and 30 on 11 narcoleptics. One normal subject was studied during wakefulness and sleep after 100 hours of continuous experimental insomnia.

RESULTS

Factors in normal sleep

Comparison of leads. We have corroborated the findings of Loomis *et al.* (1937) and Davis *et al.* (1938) that during sleep the 10 per sec. trains are more often present at the occiput and the 14 per sec. rhythm at the vertex, and that the amplitude of the 1-3 per sec. waves is greater at the vertex. But, despite such detailed differences, all potentials are present over the entire cortex. When potentials from two head regions are compared, similar patterns of delta waves are clearly simultaneous at the parietal, occipital, frontal, and temporal regions, bursting into activity and subsiding suddenly (see also

* Aided by a grant from the Rockefeller Foundation.

Loomis *et al.*, 1938). Further, whether or not the 10 per sec. rhythm is present in all regions at a particular time, all leads may show bursts at this frequency at some stages of sleep. With leads from only the occipital and frontal regions, Blake and Gerard found the 14 per sec. rhythm inconspicuous; with the vertex lead it is now clearly seen, even without specially tuned circuits. The fraction of the time during which the 14 per sec. rhythm is present increases slowly, and with fluctuations, during the early part of the night (alpha + delta period, see below), reaches a maximum in the middle portion, and declines gradually as the alpha waves again appear. The amplitude of the rhythm is lowest in the period during which the slow waves predominate (compare Davis *et al.*, 1938).

Table 1. Potentials in stages of sleep

	Amt. Alpha	Amt. Delta	Amt. 14 per sec.	Depth of sleep	Present Nomen- clature	Nomenclature of Tux- edo Park Group*
1	++	—	—	Awake	Alpha	
2	+	++	+	Light sleep	Alpha + delta (+14 per sec.)	A. Interrupted alpha B. Low voltage C. Spindles
3	—	+++	+	Deep sleep	Delta (+14 per sec.)	D. Spindles + random E. Random
4	—	—	+	Light sleep	Null (or low voltage)	B. Low voltage
5	(+)	—	(+)	Sleep to wake	Intermittent alpha	B. to A. Low voltage to interrupted alpha
6	+	—	—	Awake	Alpha (low in- tensity)	

* The nomenclature of the Tuxedo Park group represented above is the result of personal communication with Dr. E. N. Harvey and Dr. H. Davis.

Sleep stages. Loomis *et al.* (1937, 1938), using tuned and untuned circuits, have described five stages of sleep: A, alpha; B, low voltage; C, spindles; D, spindles + random; and E, random. Potential patterns corresponding with these stages have been observed in the present experiments but others are also evident. The changes of potential pattern in the course of the night seem best described in terms of the combined curves of rise and fall of each individual rhythm. Despite the large and frequent fluctuations from one potential pattern to another (Blake and Gerard, 1937; Loomis *et al.*, 1937), especially when the subject is disturbed, there is a definite slow shift through the night in potential pattern prominence. This has been evaluated for the two faster rhythms, as previously for the slow one, by averaging the dominant potentials over 5 min. periods through many nights of sleep. Curves illustrating the per cent presence of alpha, delta and 14 per sec. rhythms are shown in Fig. 1. Fluctuations lasting 2 minutes or less (due to extraneous sound stimuli, etc.) are not considered.

This set of curves permits a description of potential changes during the night. The major patterns are presented in Fig. 2. This arrangement does not conflict essentially with the stages of Loomis *et al.* (1937) and seems preferable since it is based upon the quantitative measure of per cent presence of each type of wave rather than upon the more qualitative recognition of certain potential patterns. For convenience, the generally accepted, although perhaps misleading, convention has been used of assigning a letter to each wave fre-

SCHEMA OF POTENTIALS DURING A NIGHT'S SLEEP

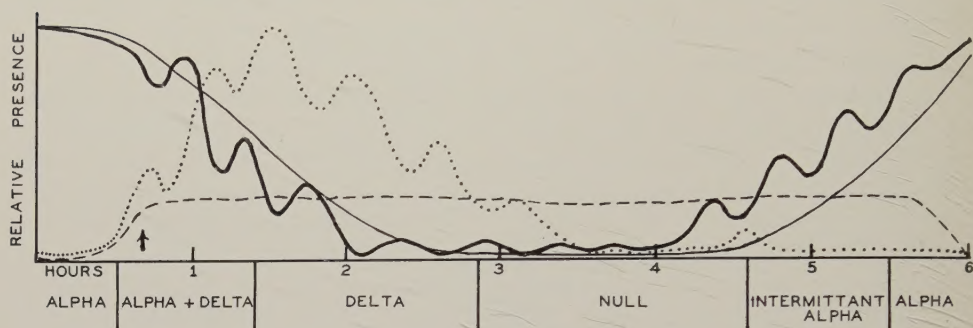


FIG. 1. Predominance of brain potentials through the night. Alpha waves (black heavy line) in per cent presence; 14 per sec. waves (dashes) in per cent presence and delta waves (dots) in extent of predominance. Oral temperature (black thin line). Below the stages of sleep are indicated. Record begun at time of retiring arrow indicates beginning of sleep. Wavy lines represent changes in waves due to shift of state of sleep. Depth of sleep by auditory response method roughly parallels delta curve.

quency. "Alpha" stands for the 10 per sec. regular rhythm usually present during the waking state. (In cases where the beta waves are the normal resting potentials, beta would replace alpha. "Alpha+delta" would be replaced similarly by "beta+delta.")* "Delta" is used for the 0.5 to 5 per sec. sometimes irregular wave dominant in deep sleep. The period of light sleep in the third quarter of the night, when delta waves have disappeared and the flat base line is broken only by an infrequent slow wave, or by a burst of alpha or beta waves, is called "null" (or low voltage) (Table 1).

The changes during diminishing sleep in the late part of the night fail to mirror those of increasing sleep in the early part in the following respects

* Personal communications from Drs. Harvey and Davis have brought up the question whether the 10 per sec. rhythm seen simultaneously with the delta rhythm in the "alpha+delta" period is the same as the 10 per sec. wave of wakefulness or whether it is the beta component which has been slowed from 25 to 10 per sec. This question cannot be answered from the present work. It seems clear that the 10 per sec. rhythm in this "alpha+delta" period is not so closely associated with consciousness as it is later in the night (see below) and may, therefore, have a different significance. On the other hand, normal "beta" subjects show a "beta+delta" stage in contrast to the more common "alpha+delta"; and notched waves at this stage in the alpha case seem to be in transition between alpha and delta.

(Fig. 2): (i). The delta component reaches a peak in the second hour of sleep, then gradually disappears in another hour or two. (ii). The delta waves first appear before the alphas disappear, producing "notched" waves (Fig. 2b; also Blake and Gerard, 1937); whereas later the deltas fade some hours before the alpha waves return, leaving an essentially flat base line. (iii). The alpha waves are 20 to 40 per cent larger and the betas more prominent and of higher frequency just before falling asleep than just after awakening. (iv). Consciousness is associated with the presence of alpha waves in the "null" period but

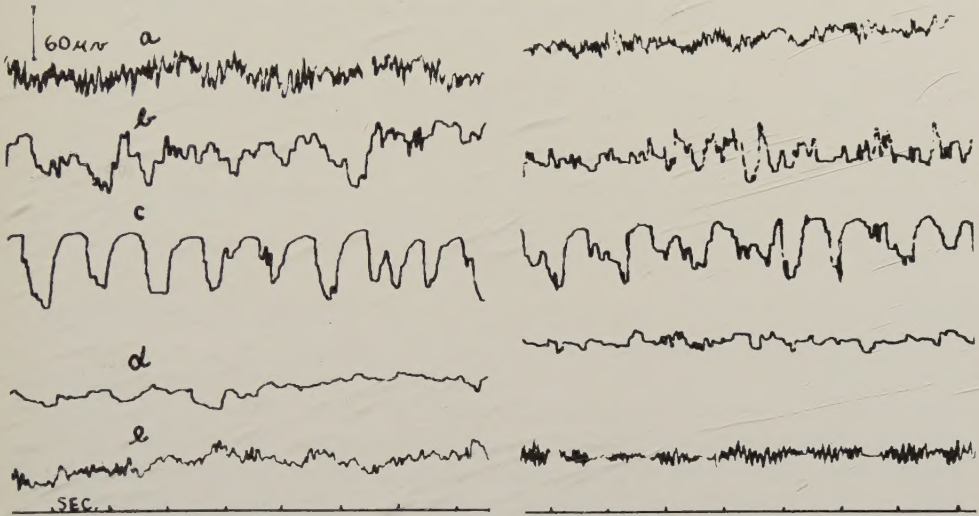


FIG. 2. Potential patterns through the night on three subjects. 1. "Alpha" rhythm of wakefulness. 2. "Alpha + delta" period of light sleep. 3. "Delta" period of deep sleep. 4. "Null" period of light sleep. 5. "Alpha" rhythm of wakefulness.

not necessarily in the earlier "alpha + delta" period. (v). Weak stimulation, such as slight movement or noise, affects brain potentials, particularly the delta rhythm, oppositely in the early and in the late period of light sleep (Loomis *et al.*, 1937; Blake and Gerard, 1937). In the "alpha + delta" stage it usually diminishes the delta waves, which emphasizes the alphas. In the "delta" and early in the "null" stages, a similar stimulus either exaggerates the slow waves already present or initiates them and also a faster rhythm, which last for a few seconds. This corresponds to the "K complex" of Loomis *et al.* (1938). Later in the "null" stage stimulation causes alpha waves to appear. It seems, then, that a stimulus which produces a shift towards lighter sleep causes the potentials present at the time to pass through those of the preceding stage before reaching the alpha waves of wakefulness.

Consciousness. The relation between consciousness and brain potentials was investigated by Davis *et al.* (1938) by having the subject squeeze a bulb when aware of having "drifted off." They found that alpha waves, which were absent during a "float," had returned between 3 and 23 sec. before the

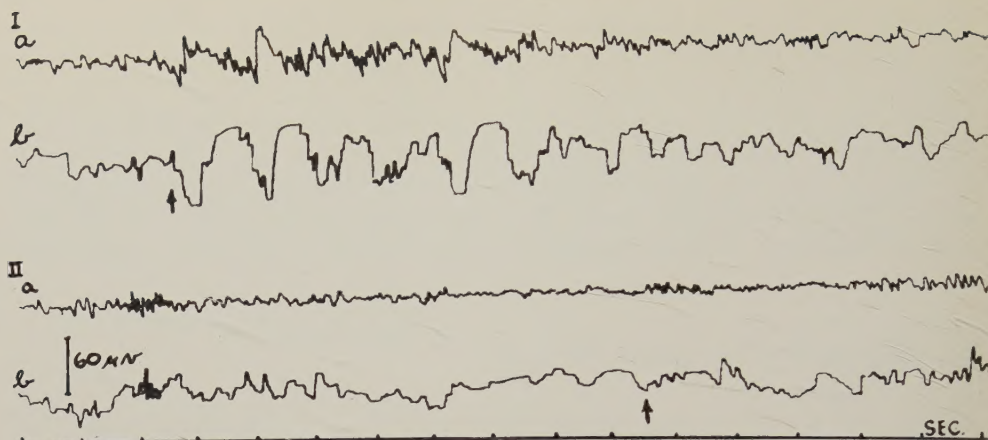


FIG. 3. Effect of slight stimulation (at arrows) on brain potential pattern: 1. As the delta waves are diminishing after deep sleep. a. Low cut-off filter in, b. No filter. 2. Late in the "null" period. a. Filtered. b. Unfiltered. Note that whereas in the first record stimulation elicits a train of delta waves, later it does not.

signal was given. We have studied the loss rather than the return of consciousness and related it to dreams and to skeletal muscle tonus.

A. *Dreams*. Loomis *et al.* (1936) at first suggested that dreams were associated with a "peculiar" slow wave, but later (1937) decided that they are not associated with any particular wave but occur in the B stage of sleep. Davis *et al.* (1938) find dreaming also in the C stage. In the present experiments, the subject was suddenly awakened while some particular wave pattern was present, and asked whether he had been asleep and, if so, whether and about what he had dreamed. Some individuals did not show sharp potential changes from one sleep level to another and others found it difficult to decide clearly whether or not sleep and dreams had been experience. In all subjects

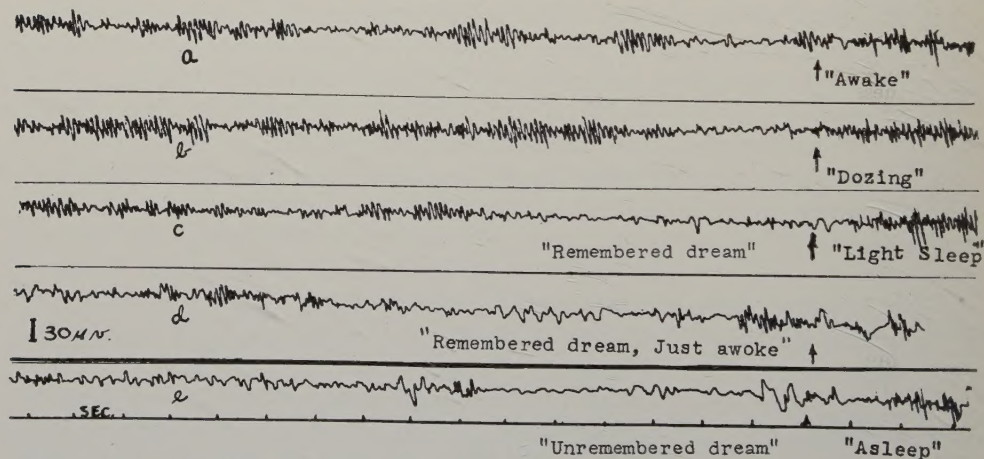


FIG. 4. Record in the "null" period of light sleep with low cut-off filter. Subject was awakened and questioned about dreaming at the arrow.

a uniform general correlation between potentials and dreams was present, but the following details are based largely upon results obtained on one young woman able to give decisive subjective reports (Table 2, Fig. 4): (i) Report, "awake." In the "null" period, the presence of alpha waves, even for one second, was invariably associated with a report of consciousness. (Fig. 4a); (ii) Report, "dozing"—some dimming of awareness and decreased sense of reality of surrounding events. This was the report when alpha waves had been absent for at least 3, average 6, sec. (Table 2). (iii) Report, "remembered

Table 2. *Brain potentials and introspection during sleep in second half of the night (one subject)*

Subjective Impressions	Duration of period with no alpha		Presence of delta	Number of queries
	Average sec.	Range sec.		
Wakefulness	0	—	—	6
Dozing	6	3-7	—	7
Remembered dream	9	2-16*	—	15
Unrecalled dream	55	6-120	+	6
Dreamless	?	?	+	Rarely occurred

* In one case there were no alpha waves for 150 sec. before the query but this is the exception; the usual figures are close to 9 sec.

dream"—light sleep with a dream clearly remembered. In nine-tenths of the trials, when alpha waves had been missing for 9 sec. (and no delta waves were present) the subject could remember a dream. (iv) Report, "unrecalled dream"—deeper sleep with a clear memory of having dreamed but no recall of content. This was the report when alpha waves had been absent, on the average, for 55 sec. Delta waves were usually present. (v) Report, "dreamless sleep"—deep sleep with no suggestion of having dreamed. This report paralleled a prominent delta rhythm, and rarely occurred in the second half of the night or early in the alpha+delta stage.

The subject was awakened at irregular intervals throughout the night in these tests and it is clear that dreaming was present most of the time, although minimal in the second quarter (delta period). The change from thinking to dreaming, with advancing sleep, seems to be less an immediate depression of mental activity than a progressive shift of attention from exteroceptive sensations towards subjective imagery. Even in deep "dreamless" sleep there is only a short period during which cortical activity is probably depressed to such an extent that the subject is not aware, on abrupt awakening, of having dreamed.

B. *Tonus*. As a test for tonus, the subject held between two fingers a light spool, which fell as the muscles relaxed in sleep. The subject was then aroused and asked whether or not he had been aware of dropping the object. The spool usually fell between 0.5 and 1.5 (average 1.1) sec. after the alpha rhythm had disappeared. The subject was then aware of its fall. Occasionally, however,

the fall was delayed until 6.5 to 25 (average 14) sec. after alpha loss, in which case the subject was unconscious of having dropped it. Tone, therefore, diminishes soon after the alpha rhythm is lost, but consciousness does not disappear for some seconds more. Subjective "dozing," shown above to follow the disappearance of alpha waves by 6 sec., is experienced between loss of tone and loss of consciousness. The subjects of Davis *et al.* (1938) similarly did not consider "floating" or "dozing" as real sleep.

Table 3. *Effect of movement on brain potential patterns*

Type of brain wave change	Duration of movement (sec.)	Duration of brain wave change (sec.)	Number of movements
$\alpha + \Delta \rightarrow \alpha$	21	14	6
$\Delta \rightarrow \alpha$	22	24	13
null $\rightarrow \alpha$	19	28*	20

* Several times changes, not included in the table, lasted for minutes and the level of sleep was permanently changed.

Motility. Blake and Gerard (1937) found that movement was regularly associated with a shift to lighter sleep. Loomis *et al.* (1937) report that "Movement may occur without a change of state (of sleep) and a change of state without movement, but frequently movement is immediately followed by a change of state upward, occasionally downward. . . ." In 90 per cent of movements in which muscle tension lasted over 5 sec. (80 instances now analyzed in detail) brain potentials shifted to a pattern of a lighter sleep; in 10 per cent they did not change, mainly in the null period. There was no shift towards a deeper level. (A transient increase in synchrony of delta waves in the third quarter of the night is evidence of decreased depth of sleep; cf. above.) Average values for the duration of movement (about 20 sec.) and the direction and duration of potential changes (progressively longer from the "alpha + delta" through the "null" stages) are given in Table 3. Occasionally sleep would remain lighter for over an hour following a movement. In some 5 per cent of all observations, alpha waves appeared before movement occurred, suggesting that extero- or interoceptive stimuli were responsible for the change; and even when brain and muscle activity were simultaneous such stimuli may have lightened sleep sufficiently to permit proprioceptive reflexes to break through. Certainly after movement is initiated the new proprioceptive barrage would tend to cause awakening.

Temperature. Alpha frequency varies with temperature in the waking subject (Hoagland, 1936; Jasper, 1936). The diurnal temperature change, $0.5^{\circ}\text{C}.$, would only account for a frequency change between 10 and 9.7 per sec., assuming the μ value of 7000–8000 cal. (Hoagland). There is a 10–20 per cent slowing of the alpha rhythm with the onset of sleep (Davis *et al.* 1938) and a further diminution through the night, in close parallel with the amount of tremor (Jasper, 1938). We find the per cent presence of alphas parallels the temperature curve during sleep rather closely as both fall to,

and maintain, a low level, while only late in the "null" period does the alpha curve rise in advance of that for temperature (Fig. 1). This correlation is reasonable since the alpha rhythm is associated closely with tonus. (See above; also Jasper, 1938.) A decline in muscle tone should decrease body temperature gradually; an increase in tone should raise temperature, but not so quickly as it restores the alpha rhythm. Both the alpha rhythm presence and the temperature are lower in the morning after waking than they were the previous evening.

Abnormal conditions

Experimental insomnia. Behavior and potentials during prolonged insomnia and subsequent sleep have been studied (Kleitman, 1923; Blake and

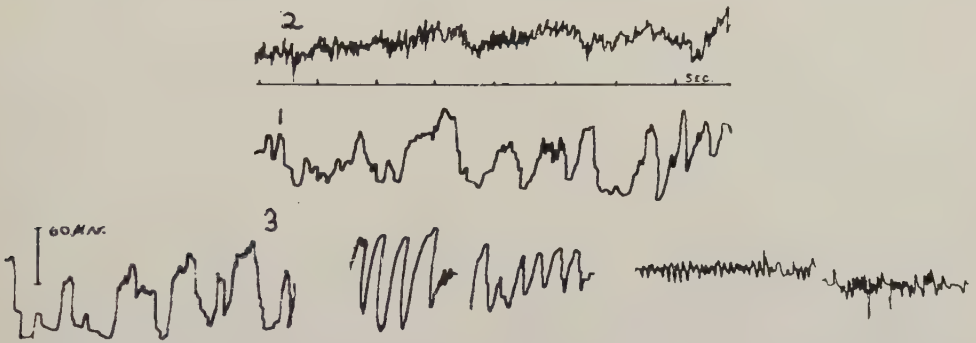


FIG. 5. Record of subject with 100 hours' insomnia. 1. "Normal" record. 2. When subject was trying to concentrate on counting. 3. Several regular potentials appearing within a 5 min. period.

Gerard, 1937). Observations have now been made on one subject, of the dominant alpha type, after 100 hours of insomnia. (Benzedrine was taken at intervals; Kleitman, unpublished.) Slow waves predominated with the subject recumbent, even though talking or with the eyes open, and they disappeared only when he made an extreme effort to concentrate. The 3-5 per sec. rhythms with 14 per sec. superimposed were the most common potentials; but all varieties of slow waves appeared and in no regular sequence with deepening sleep. Muscular tone was so low that even with distinct effort the spool was never held more than 15 sec., and usually it was dropped immediately.

The subject was never aware of having slept and thought he answered every question, but actually a strong auditory stimulus was often required to arouse him to the point of responding, which shows failure to differentiate sleep from wakefulness. To check the maximum duration of wakefulness, the subject counted as long as he could. This required intense concentration and was paralleled by a great discharge of beta waves which displaced the delta rhythm. Even with this effort, consciousness was lost at a count between 3 and 10 and, in about as many seconds, the potentials drifted back to the usual slow ones. Possibly most striking, was the play of many regular rhythms.

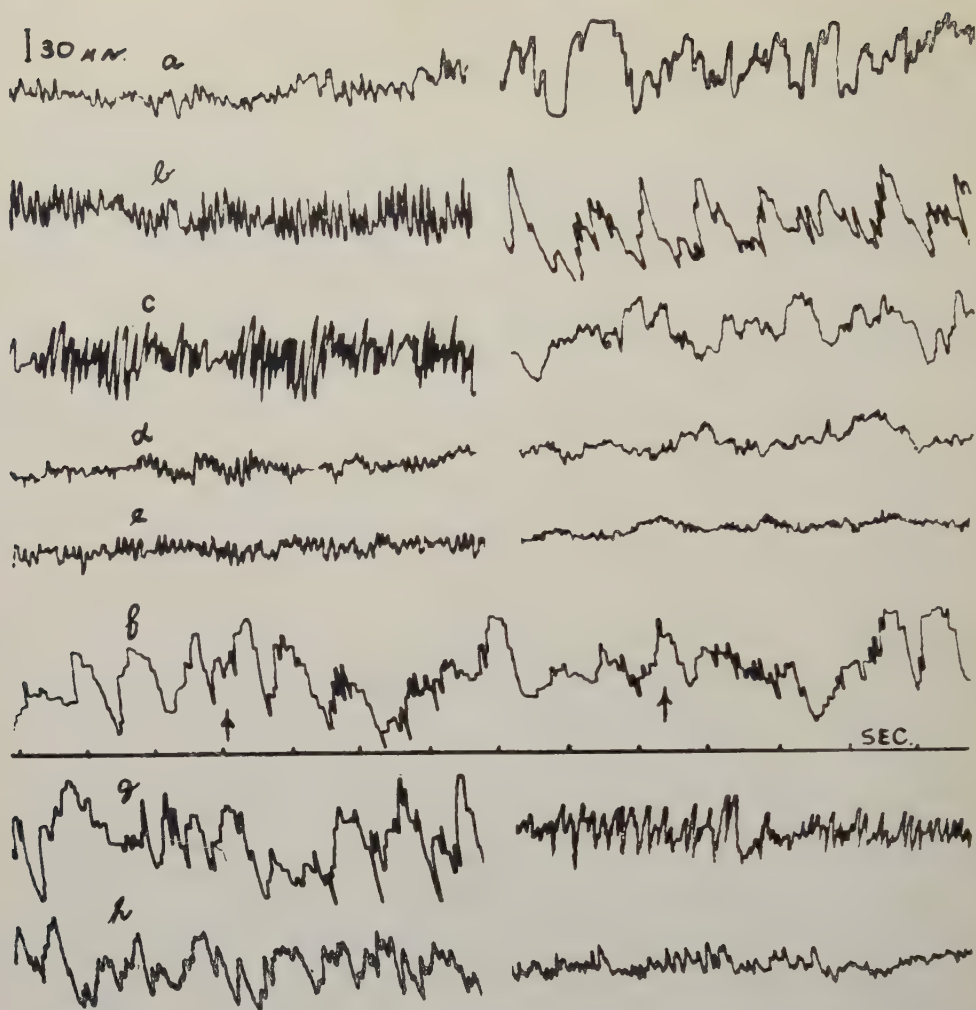


FIG. 6. a, b, c, d, e. Characteristic records of potentials from narcoleptics: 1. Lying down. 2. Sitting up. f. Response of narcoleptic to query in spite of presence of delta waves. g, h. Record of two narcoleptics lying down. Left. Before benzedrine. Right. One hour after 10 mg. benzedrine orally.

Within a five minute period: 1, 2, 3, 10 and 14 per sec. rhythms were clear, besides the high frequency beta discharge (Fig. 5). The genesis of these rhythms will be discussed later.

Narcolepsy. Eleven narcoleptic patients, male and female, of varying ages, were studied for four months during day and night sleep. Two had an associated obesity and one was an alcoholic.* In 10 of the cases a good alpha predominated in the sitting position; but on lying down this was always markedly diminished, and was usually replaced by large delta waves. The change oc-

* We are indebted to Dr. Walter Adams for the opportunity to study these patients. He will report elsewhere on the clinical aspects of this group of narcoleptics.

curred simultaneously in occipital, frontal, parietal and temporal leads, as in normal sleep. Delta frequencies from 0.5–5 per sec. appeared irregularly in each case (Fig. 6), much as in deep normal sleep. This is in contrast to the normal pattern of rest, in which the alpha rhythm persists through hours in the recumbent position, and even to that of day-time naps, in which the alpha rhythm is often merely depressed and delta waves are uncommon (Blake and Gerard, 1937). The correlation between depth of sleep, as determined by the auditory response method, and type of potentials was normal. Some patients characteristically slept deeply, others lightly, but all showed wide variations in sleep level. Since the changes between wakefulness and sleep are marked so clearly in these patients by changes in brain potentials, the electrical method may prove valuable for the objective determination of the frequency and duration of the narcoleptic attacks. It should be particularly useful in stuporous patients for discriminating sleep from simple unresponsiveness.

Drugs (a) *Benzedrine*. This drug is reported (Davidoff and Reifstein, 1937) to increase wakefulness, excitation, mental activity, metabolism, and sympathetic stimulation, and was found (Blake and Gerard, 1937) to diminish the large delta waves present during sleep after prolonged insomnia. In 10 experiments, benzedrine sulphate (10 mg.) did not obviously affect the frequency or amplitude of the waking alpha rhythm, when psychological factors were controlled. During sleep, however, the delta potentials were diminished in duration and amplitude. This was particularly marked in narcoleptics (Fig. 6).

(b) *Alcohol*. Mullin *et al.* (1937) reported that alcohol increased depth of sleep during the first part of the night and decreased it during the second half. This has been confirmed (4 experiments) and a parallel change in potentials demonstrated, delta waves being accentuated early in the night, alpha waves later. Possibly the early peripheral vasodilatation, and consequent lowering of body temperature, is one factor favoring the early deep sleep.

DISCUSSION

If one defines sleep in terms of loss of consciousness (Kleitman, 1929; Hess, 1932), the light sleep of day-time naps offers a simple case for study of the essential changes. Here the constant alteration is a diminution of the alpha rhythm. Delta waves, or the 14 per sec. rhythm, may or may not appear. Loss of consciousness and of alpha rhythm are related and the alpha rhythm, further, is associated with muscle tone, the two decreasing together. Kleitman (1929) has emphasized the importance of diminution in proprioceptive and other afferent stimuli in inducing sleep and suspending consciousness, so the close relation of alpha waves to both tonus and awareness is significant.

The work of Bremer (1935, 1937) likewise emphasizes the importance of diminished afferent impulses in producing the cortical potential changes of sleep. In the cat, mesencephalic section of the brain stem, barbiturate narcosis, and sleep are all associated with cortical waves of decreased frequency

and increased amplitude. Several workers (Jasper, 1937; Gerard, 1936; Blake and Gerard, 1937) have emphasized the relation of neural excitation level and wave frequency, the two rising and falling together. The present findings with a stimulant, benzedrine, and a depressant, alcohol, fit this picture. On this basis, diminished afferent impulses (perhaps most important, the proprioceptive) playing upon the brain, allow cortical excitation to subside with the gradual loss of consciousness and slowing of potentials. The beta waves retard to 14 per sec. spindles, as Jasper (1937) has shown; and the alpha waves are replaced by, or perhaps are changed into, the slow deltas (Blake and Gerard, 1937). Certainly the appearance of many distinct frequencies within a few minutes, after a prolonged insomnia, suggests that the same cortical neurones can beat at many rates; and the demonstration (Libet and Gerard, 1938 and unpublished) that a few homogeneous cells in the isolated frog olfactory bulb can be made to assume regular rhythms from 1 to 50 per sec. by controlling the excitation level, strongly supports such an interpretation.

It remains uncertain whether the slowed cortical rhythms of sleep are a direct consequence of lowered afferent bombardment or are secondary to a decreased cell metabolism which follows the lowered excitation level. Certainly sensory impulses increase brain heat production (Serota and Gerard, 1938) and, conversely, in sleep brain temperature falls (Serota, 1939). It is also not clear what the role of subcortical centers may be. The fairly simultaneous change in waves over most of the cortex would be in accord with a thalamic or hypothalamic control; and much evidence for such "sleep" centers exists (Hess, 1932; Ranson, 1934; Bard, 1928; Serota, 1939). The cortical changes could, of course, result from a shutting of the thalamic gateway to sensory impulses as well as from a failure to initiate them at peripheral receptors. The excessive somnolence of narcolepsy, associated with pathology in the diencephalon, may well depend on such a "blockade"; that following prolonged insomnia is more probably compounded from both these factors and a direct fatigue depression of cortical neurones as well.

Interpretations of the findings concerned with dreaming is partly beyond the scope of this paper, especially in view of the vast psychiatric literature dealing with the dynamic properties of dreams. We have shown that a subject abruptly awakened, almost at any time during the night, can recall having dreamed; the longer the immediately preceding period with no alpha waves, the less is the recall, and when this period is about a minute (especially if delta waves are present), there is no trace of a dream's having been in progress. This largely excludes the possibility that, in the other states, the dream ran its course as a flash while the subject was actually in the process of waking; presumably dream consciousness, like waking consciousness, blurs and fades progressively as the activity of the cortical neurones falls to lower and lower levels. The conclusion that delta waves appear only in complete unconsciousness, such as coma, narcosis, and epilepsy, as well as dreamless sleep, is forbidden by their presence in drowsing narcoleptics.

Finally, the sequence of changes in passing from wakefulness to deep sleep

and back to wakefulness is of interest. Muscle tone decreases first, then sharp awareness is replaced by dozing or actual dreaming but the subject can still take cognizance of events (dropping a spool, having dreamed), and finally a dreamless oblivion of deep sleep is reached. Disturbances during sleep—external or proprioceptive or perhaps even the building up of excitation in the brain itself—cause a shift towards a lighter state. This is more prolonged when the disturbance occurs late in the night than when it is early and so parallels other signs of asymmetry during a night's sleep. Thus: delta waves appear before the alphas are gone during deepening sleep, but disappear before the alpha waves return during the "null" period; in the descending phase, stimuli convert delta waves to alphas, while in the ascending stage they first initiate delta waves and start alphas only if actually arousing the sleeper; and the alphas just after awakening are feebler than just before going to sleep.

This asymmetry could be accounted for by a combination of two factors. The "fatigued" cortical cells easily fall to a low level of activity when afferent impulses decrease. As they become "rested," sleep lightens even without increased stimulation; and any stimuli that do occur then are relatively more effective than earlier ones.

SUMMARY

1. Minute to minute fluctuations in brain potentials through the night are superimposed on a gradual trend from hour to hour. This latter is compared with the sleep stages described by others. The potentials are present simultaneously over much of the cortex. In sequence, the patterns are: alpha + delta, delta, null, intermittent alpha.

2. During increasing sleep depth, early in the night, delta waves appear before alpha waves are gone; while later, during diminishing depth of sleep, the deltas disappear before the alphas return. In other respects also, potentials of the rising sleep phase do not mirror those of the falling phase.

3. Subjective reports of sleep and dreams can be correlated with potential patterns, sometimes quite sharply.

4. Movement is accompanied by a shift of potentials towards lighter sleep in nine-tenths of the present cases, by no change the remaining times.

5. Hypersomnia, due to prolonged voluntary insomnia or to narcolepsy, is associated with delta waves at relatively higher levels of consciousness than in normal sleep.

6. A stimulant drug, benzedrine, diminishes delta waves; a depressant, alcohol, enhances them.

7. These findings are discussed in relation to theories of sleep and the source of cortical potentials.

* * * *

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REFERENCES

ADAMS, W. Unpublished.

BARD, P. A diencephalic mechanism for the expression of rage with special reference to the sympathetic nervous system. *Amer. J. Physiol.*, 1928, 84: 490-515.

- BLAKE, H., and GERARD, R. W. Brain potentials during sleep. *Amer. J. Physiol.*, 1937, 119: 692-703.
- BREMER, F. Cerveau "isolé" et physiologie du sommeil. *C. R. Soc. Biol., Paris*, 1935, 118: 1235-41.
- BREMER, F. Différence d'action de la narcose éthérique et du sommeil barbiturique sur les réactions sensorielles acoustiques du cortex cérébral. Signification de cette différence en ce qui concerne le mécanisme du sommeil. *C. R. Soc. Biol., Paris*, 1937, 124: 848-52.
- DAVIDOFF, E., and REIFENSTEIN, E. C. Stimulating action of benzedrine sulphate; comparative study of response of normal persons and of depressed patients. *J. Amer. med. Ass.*, 1937, 108: 1770-76.
- DAVIS, H., DAVIS, P., LOOMIS, A. L., HARVEY, E. N., and HOBART, G. Human brain potentials during the onset of sleep. *J. Neurophysiol.*, 1938, 1: 24-38.
- GERARD, R. W. Factors controlling brain potentials. *Cold Spr. Harb. Symp.*, 1936, 4: 292-304.
- HESS, W. R. The autonomic nervous system, *Lancet*, 1932, 2: 1199-1201; 1259-61.
- JASPER, H. H. Electrical signs of cortical activity. *Psychol. Bull.*, 1937, 34: 411-81.
- JASPER, H. H., and ANDREWS, H. L. Brain potentials and voluntary muscle activity in man. *J. Neurophysiol.*, 1938, 2: 87-100.
- KLEITMAN, N. Studies on the physiology of sleep. I. The effect of prolonged sleeplessness on man. *Amer. J. Physiol.*, 1923, 66: 67-92.
- KLEITMAN, N. SLEEP. *Physiol. Rev.*, 1929, 9: 624-65.
- KLEITMAN, N. New methods for studying motility during sleep. *Proc. Soc. exp. Biol., N.Y.*, 1932, 29: 389-91.
- LOOMIS, A. L., HARVEY, E. N., and HOBART, G. Cerebral states during sleep, as studied by human brain potentials. *J. exp. Psychol.*, 1937, 21: 127-44.
- LOOMIS, A. L., HARVEY, E. N., and HOBART, G. Distribution of disturbance-patterns in the human electroencephalogram with special reference to sleep. *J. Neurophysiol.*, 1938, 1, 413-30.
- LIBET, B., and GERARD, R. W. Chemical control of potentials of the isolated frog brain. *Amer. J. Physiol.*, 1938, 123: 128 P.
- MULLIN, F. J., KLEITMAN, N., and COOPERMAN, N. R. Studies on the physiology of sleep. X. The effect of alcohol and caffeine on motility and body temperature during sleep. *Amer. J. Physiol.*, 1933, 106: 478-87.
- OFFNER, F., and GERARD, R. W. Push-pull resistance coupled amplifiers. *Rev. Sci. Instr.*, 1937, 8: 20-21.
- OFFNER, F. A high speed crystal ink writer. *Science*, 1936, 84: 209-10.
- RANSON, S. W. The hypothalamus: Weir Mitchell Lecture. *Trans. Coll. Phys., Philad.*, 1934, 2: 222-42.
- SEROTA, H. M. and GERARD, R. W. Localized thermal changes in the cat's brain. *J. Neurophysiol.*, 1938, 1: 115-24.
- SEROTA, H. M. Temperature changes in the cortex and hypothalamus during sleep. *J. Neurophysiol.*, 1939, 2: 42-47.

